EPA/822/R-98/005
July 1998

Ambient Water Quality Criteria for the Protection of Human Health: 1,3-Dichloropropene (1,3-DCP)

NOTE TO READER

The Agency is intending to develop streamlined criteria documents which focus on critical toxicological and exposure related studies only. This is a departure from the past format in which all existing toxicological and exposure studies were presented and evaluated in the 1980 criteria documents, with equal emphasis placed on exposure, pharmacokinetics, toxicological effects and criterion formulation. Due to limited resources and a need to update criteria as quickly as possible, U.S. EPA has decided to develop more abbreviated versions of criteria documents with an emphasis on using existing risk assessments (on IRIS or other U.S. EPA health assessment documents) where available and still relevant, and focusing to a greater extent on pertinent exposure and toxicological studies which may influence the development of a criterion (e.g., critical effects studies which form the basis of RfD development or cancer assessment). The U.S. EPA will continue to conduct a comprehensive review of the literature for the latest studies, but will not provide a summary or an evaluation of those studies in the criteria documents which are deemed less significant in the criteria development process. Where there is a significant amount of literature on an area of study (i.e., pharmacokinetics), the U.S. EPA, to the extent possible, will reference the information or cite existing documents (e.g., IRIS or other existing U.S. EPA risk assessment documents) which discuss the information in greater detail.

The overall objective of this change in philosophy is to allow the U.S. EPA to update 1980 AWQC at a greater frequency, while still maintaining the scientific rigor which the U.S. EPA requires when developing an AWQC. The U.S. EPA believes these "new" criteria documents will be just as informative as previous criteria documents and will continue to serve as the key scientific basis for State and Tribal standards. The U.S. EPA also believes the documents will provide the necessary scientific content and scope to allow a State or Tribe to come to an appropriate technical and/or policy decision with regard to water quality standards setting decisions.

The U.S. EPA requests that commenters identify any relevant information missing from this criteria document which may result in different criteria calculations or scientific interpretation. EPA also requests comments on the change in criteria document format. This criteria document has undergone extensive external peer review.

1. BACKGROUND

Under the previous methods for setting ambient water quality criteria established in 1980, criteria for dichloropropenes as a group were set based on non-carcinogenic effects (USEPA, 1980). The criterion to protect against ingestion of water and aquatic organisms was set at 87 µg/L, and the criterion based on ingestion of contaminated aquatic organisms alone was 14,100 µg/L. Criteria for 1,3-dichloropropene (1,3-DCP) were set under the National Toxics Rule (USEPA, 1992a) at 10 µg/L for ingestion of water and aquatic organisms and at 1,700 µg/L for ingestion of aquatic organisms only.

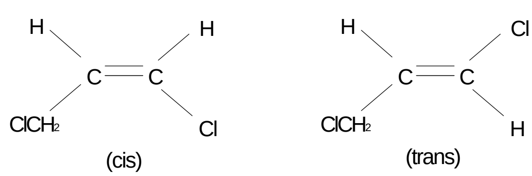
This criteria document updates national criteria for 1,3-DCP using new methods and information described in the *Federal Register* (*FR*) notice (USEPA, 1998a) and Technical Support Document (TSD) (USEPA, 1998b) to calculate ambient water quality criteria. These new methods include updated approaches to determine dose-response relationships for both carcinogenic and non-carcinogenic effects, updated information for determining exposure factors (e.g. values for fish consumption), new exposure assumptions used in the calculation, and new procedures to determine bioaccumulation factors.

In addition to new methods for deriving AWQC values, new data on toxicity, exposure, and bioaccumulation of 1,3-DCP are also included in the criteria calculation. The criterion proposed to protect against ingestion from water and aquatic organisms (based on carcinogenic effect) is 0.34 µg/L, and the criterion to protect against ingestion of aquatic organisms and incidental water exposure is 14 µg/L. These values are calculated based on a lifetime risk of 10^{-6}. The calculation is based on adults in the general population.

The following sections include the toxicological, exposure, and bioaccumulation factor evaluations, the calculation of the criteria, and a discussion of site-specific adjustments to the criteria.

2. CHEMICAL NAME AND FORMULA

The AWQC is being derived for 1,3-dichloropropene (CAS No. 542-75-6). The chemical formula is $C_3H_4Cl_2$, and the structural formulae are:



1,3-Dichloropropene (approximately 46% trans, 42% cis)

Synonyms

Synonyms include the following: 1,3-D; 1,3-dichloropropylene; Telone; Telone II; 3-chloroallyl chloride; 3-chloropropenyl chloride; alpha, gamma-dichloropropylene; gamma-chloroallyl chloride.

Physical and chemical properties (USEPA, 1989)

Chemical Formula	$C_3H_4Cl_2$
Molecular Weight	110.98 (pure isomers)
Physical State (25ºC)	Pale yellow to yellow liquid
Boiling Point	about 104ºC (104.3ºC, cis; 112ºC, trans)
Density (25ºC)	1.21 g/ml
Vapor Pressure (25ºC)	27.3 mm Hg
Specific Gravity	about 1.2 (20/20ºC)
Water Solubility (25ºC)	0.1 to about 0.25% (1 to 2.5 g/L) reported; miscible with most organic solvents
Log Octanol Water Partition Coefficient	1.76
Conversion Factor (25ºC)	1 mg/L = 220 ppm; 1 ppm = 4.54 mg/m^3

3. SUMMARY OF PHARMACOKINETICS

Toxicity studies indicate that 1,3-DCP[1] is absorbed from the respiratory tract and gastrointestinal system (Hutson et al., 1971; Deitz et al., 1985; Stott and Kastl, 1986; Waechter and Kastle, 1988). There are no data available on the dermal absorption of 1,3-DCP. Absorption through the skin can be inferred, however, from dermal toxicity studies using rabbits (Lichy and Olson, 1977). By the oral route, approximately 90 percent or more of the administered dose is absorbed in rats (Hutson et al., 1971; Deitz et al., 1985; Waechter and Kastl, 1988).

The vast majority of an orally or inhalation administered dose of 1,3-DCP appears to be metabolized and excreted in the urine (ranging from 50 to 80%). A smaller amount is excreted in the expired air (approximately 14-26%) and in the feces (approximately 14-18%). After 48 hours of dosing, only minor amount of the administered dose (2-6%) remained in the carcass of rats and mice. No major sex differences were noted. Absorbed DCP is metabolized mainly by conjugation with glutathione, followed by further metabolism to a mercapturic acid, and is excreted in the urine as an acetylated cysteine derivative (*N*-acety-S-[(3-chloroprop-2-enyl]cysteine) (Climie et al., 1979; Fisher, 1988; Fisher and Kilgore, 1988p; Waechter and Kastl, 1988). In addition to the mercapturic acid of 1,3-DCP, its sulfoxide is also identified as a urinary metabolite (Waechter and Kastl, 1988).

[1]Although 1,3-dichloropropene is usually abbreviated as 1,3-DCP throughout this document, some studies do not specify the form of dichloropropene. Therefore, in some places, the chemical is abbreviated as DCP.

Dietz et al. (1985) and Waechter and Kastl (1988) both found that after oral administration of 1,3-DCP, very small amounts of the chemical were found in any tissue of either rats or mice, but the highest concentrations were found in the non-glandular stomach.

4. TOXICOLOGICAL BASIS FOR CRITERIA

4.1 Noncancer Data and Previous Evaluation

4.1.1 Human Data

In humans, one death has been found to be related to ingestion of 1,3-DCP (Gosselin et al., 1976). Symptoms observed included abdominal pain, vomiting, muscle twitching, and pulmonary edema. In addition, Venable et al. (1980) studied 64 male workers exposed to 3 carbon compounds (one being 1,3-DCP) to determine effects on fertility. The control group consists of 63 male workers not exposed to industrial chemicals for at least 5 years before the study. No significant differences were found in sperm counts or percent of normal sperm between the exposed and unexposed groups.

4.1.2 Animal Data

Several acute toxicity studies by oral, inhalation, and dermal routes have been performed. The oral data are summarized here. Oral LD_{50}s range from 140-710 mg/kg in rats and 300-640 mg/kg in mice (Torkelson and Rowe, 1981; Hine et al., 1953; Toyoshima et al., 1978a,b; Jones and Collier, 1986).

Oral studies using experimental animals include a sub-chronic gavage study (Til et al., 1973) and a chronic gavage study (NTP, 1985).

Til et al. (1973) administered 0, 1, 3, 10 or 30 mg/kg of Telone II (78.5% 1,3-DCP) by gavage in propylene glycol, 6 days/week for 13 weeks to 10 albino Wistar rats/sex/dose level. No effects on body weight, food consumption, or clinical chemistry tests (hematology, serum enzyme activities, and urinalysis) were found at any dose level. Histopathologic examinations were performed on many tissues and organs in the control and the high dosed group, but only on liver and kidney in the 1, 3, and 10 mg/kg groups. Relative kidney weights were increased in both the males and the females treated with 30 mg/kg and in the males treated with 10 mg/kg. No adverse effects were reported for the 1 or 3 mg/kg dose group. A NOAEL of 3 mg/kg and a LOAEL of 10 mg/kg (6 days/week) were identified for kidney effects in rats.

The National Toxicology Program (NTP) (1985) studied the chronic toxicity and carcinogenicity of Telone II in F344 rats and B6C3F1 mice. Groups of 52 rats/sex were administered Telone II (89% 1,3-DCP) via gavage with doses of 0, 25, or 50 mg/kg of Telone II in corn oil, 3 times/week for 104 weeks. Groups of 50 mice/sex/dose were similarly treated with 0, 50, or 100 mg/kg of Telone II for 104 weeks. No differences in survival were found in either male or female treated rats; however, in the mice, survival of the high-dose females was significantly less than that of the controls. Body weights of the high-dose male rats were depressed 5% relative to those for low-dose and/or control male rats. In the mice, body weights of dosed groups were 6-22% lower than those of controls at the start of the study; although the differential in body weight decreased to 5-9% by the end of the study. Rats showed increased incidences of basal cell or epithelial hyperplasia of the fore stomach in both sexes at both treatment levels, edema of the urinary bladder in both sexes at the highest treatment level, and nephropathy in females at both treatment levels. Non-carcinogenic effects in mice included increased incidences of hyperplasia of the fore stomach in high-dose female mice, a dose-related increase in hydronephrosis in female mice, and dose-related increased incidence of epithelial hyperplasia of the urinary bladder in both sexes at both treatment levels (NTP, 1985). A NOAEL or LOAEL was not identified in rats or mice in this study.

An oral RfD of 3×10^{-4} mg/kg-day has been established for 1,3-DCP and verified in 1987 based on increased kidney weights in albino rats from the 90-day sub-chronic feeding study (IRIS, 1996). The RfD was determined by applying a total uncertainty factor of 10,000 (for intra- and interspecies differences in toxicity, for using a study of sub-chronic duration, and for deficient data base) to a NOEL of 3.0 mg/kg-day. Confidence in this RfD is low, because the study on which the RfD is based is of low quality and of short duration, and the existing data base is assigned a low confidence rating by EPA.[2]

4.2 Cancer Evaluation and Available Data

A brief evaluation of whether 1,3-DCP is a potential carcinogen is discussed below. This evaluation considers data in humans and animals by all exposure routes.

4.2.1 Human Data

Human data are inadequate to establish a basis for carcinogenicity. The human data on 1,3-DCP includes three cases of cancer that developed after accidental exposure to 1,3-DCP (Markovitz and Crosby, 1984). The cancers were two malignant lymphomas and one acute myelomonocytic leukemia. These clinical reports are insufficient to draw any conclusion.

4.2.2 Animal Data

Data on carcinogenicity are available from oral, inhalation, and subcutaneous routes, and by skin painting of 1,3-DCP in mice and rats.

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[2] EPA's Office of Pesticide Programs has recently adopted a new RfD of 0.025 mg/kg-day for 1,3-DCP, based on a chronic study in rats with a NOEL of 2.5 mg/dg-day and an UF of 100. The LOEL for the study was identified as 12.5 mg/kg-day, based on decreased body weight gain and an increased incidence of basal cell hyperplasia of the nonglandular mucosa of the stomach.

One oral carcinogenicity study has been conducted (NTP, 1985), and it showed a positive response in both rats and mice. F344 rats were administered 0, 25, or 50 mg/kg of Telone II (88 to 89% 1,3-DCP, 2.5% 1,2-dichloropropane, 1.5% trichloropropane isomer, 1% epichlorohydrin, and 7.5% unspecified) in corn oil by gavage, 3 times a week for 104 weeks. A total of 52 rats/sex were used for each dose group, with 25/sex per group served as interim sacrifice. Statistically significant increases in incidence of the following tumors were observed at the high dose group by pairwise comparisons with controls: (1) fore stomach squamous cell papillomas in males and females; (2) combined fore stomach squamous cell papillomas and carcinomas in males; and (3) liver neoplastic nodules (hepatocellular adenoma) and/or carcinomas in males. In the same study (NTP, 1985), male and female B6C3F1 mice (50/sex/group) were also gavaged with 0, 50, or 100 mg/kg Telone II in corn oil. The results of the study in male mice were confounded by excessive mortality from myocarditis in controls. The survival of female mice was lower in the high dose group than in the controls. Significantly elevated incidences of the following tumors were observed either at the high dose group or at both dose groups tested: (1) fore stomach squamous cell papillomas or papillomas and carcinomas combined in males and females, (2) urinary bladder transitional cell carcinomas in males and females, and (3) lung adenomas and adenomas and carcinomas combined in males and females.

The Potential Influencing Effects of the Two Stabilizers Added in the NTP (1985) Study

As discussed by NTP (1985), the material tested in the NTP (1985) study contains primarily isomers of 1,3-DCP. The added stabilizers or impurities present in Telone II are epichlorohydrin (1%) and 1,2-dichloropropane (2.5%). Both chemicals have been shown to be carcinogenic. 1,2-Dichloropropane caused an increased incidence of hepatocellular adenomas (in male and female mice) and a marginally increased incidence of adenocarcinomas of the mammary gland in female mice. These neoplasms are different from the principal neoplastic lesions caused by Telone II. Epichlorohydrin, like Telone-II, caused an increased incidence of fore stomach tumors in the rat. However, Telone II also induced other tumor types. Thus, the impurities may contribute partially to the occurrence of fore stomach tumors, but not all tumor types.

An inhalation carcinogenicity study was performed with technical grade 1,3-DCP (92% pure) by Lomax et al. (1989) using F344 rats and B6C3F1 mice. Groups of rats and mice (50/sex/group) were exposed to 0, 5, 20, or 60 ppm 1,3-DCP for about two years. Additional groups of rats and mice were similarly exposed for 6 or 12 months. No clinical signs of toxicity or significant differences in survival were found in any group of exposed animals. No significant increased incidence of tumors was found in treated rats. However, in male mice at the 2-year sacrifice, a statistically significant increase in the incidence of bronchioalveolar adenoma (a benign lung tumor) was found at 60 ppm. A significant positive statistical trend was also found. Tumors were not found in treated female mice.

Several cancer studies were performed by Van Duuren et al. (1979). The investigators administered weekly subcutaneous injections of *cis*-1,3-DCP to 30 female HA:ICR mice at a dose of 3 mg/week. After 538 days, six of the mice had local sarcomas and no distant tumors were observed. In a second protocol, Van Duuren et al., (1979) also studied the tumor-initiating and carcinogenic potential of 1,3-DCP when applied to the skin of female HA:ICR mice. No significant differences in local or distant tumors were found between the treated and control animals when 122 mg of 1,3-DCP was administered. In a third protocol, 1,3-DCP was tested as a complete carcinogen after administration of 122 mg by dermal administration three times/week for up to 589 days, three out of 30 treated animals (but none of the 30 controls) had papillomas and/or carcinomas of the skin.

4.2.3 Other Information Relevant to the Cancer Evaluation

4.2.3.1 Mutagenicity

1,3-DCP was a direct mutagen for several *Salmonella* strains. It was mutagenic for reverse mutation in *Salmonella typhimurium* strains TA100 and TA1535 both with and without metabolic activation and in strain TA98 without metabolic activation (Haworth et al., 1983; NTP 1985; Stolzenberg and Hine, 1980; Sudo et al., 1978; Vithayathil et al., 1983). Although a few studies have not found positive mutagenic activity, evidence of mutagenicity has been found in the following studies: positive findings in the sex-linked recessive lethal assay with *Drosophila melanogaster* (NTP, 1985; Valencia et al., 1985) and the findings by several investigators that both the *cis*- and *trans*- isomers of 1,3-DCP were mutagenic in several strains of *Salmonella* (Sudo et al., 1978; NTP, 1985; Haworth et al., 1983; Vithayathil et al., 1983; Creedy et al., 1984; Neudecker et al., 1977; De Lorenzo et al., 1977). Additional studies in mammalian cell cultures also indicate that DCP is mutagenic (NTP, 1985).

4.2.3.2 Structural Analogue Data

A structurally related analogue, vinyl chloride, is a known carcinogen. Vinyl chloride has been shown to induce multiple tumors in humans and animals (IARC 1974, 1979, 1987).

4.2.3.3 Mode of Action

The current scientific consensus is that there is virtually complete correspondence between the ability of an agent to have a direct DNA activity and carcinogenicity. The data on short-term studies, as a whole, support a mutagenic mode of action. Based on the assumption of a mutagenic mode of action, and lack of information supporting a nonlinear approach for this chemical, a default low dose linearity is assumed. See further discussion of the use of mode of action data in USEPA 1996a, and USEPA 1998a, 1998b.

4.2.4 Previous Cancer Risk Evaluation

An assessment of carcinogenicity for 1,3-DCP has been done by EPA (USEPA, 1997). The tumor data of the female mice and male rats in the NTP gavage study (1985) were used as the basis to determine the cancer potency factor. The risk assessment procedures are in keeping with the EPA cancer risk assessment approach contained in the 1986 guidelines (USEPA, 1986). For this assessment, 1,3-DCP was classified as a probable human carcinogen (Class B2). An oral cancer potency factor of 1.7 x 10^{-1} $(mg/kg\text{-}day)^{-1}$ was established using the linearized multistage procedure and extra risk.

In developing the risk estimates, two separate slope factors were calculated, one from the female mice, and one from the male rats. A slope factor of 0.17 per (mg/kg-day) was obtained for 1,3-DCP from a geometric mean of the two slope factors.

In the female mice, the tumors in the bladder, lung, and fore stomach are combined. The pooled incidence counts mice with any of the three tumor types, and an oral slope factor of 0.23 per mg/kg-day was calculated. Likewise, in the male rats, the tumors in the liver, and fore stomach are combined; the pooled incidence counts male rats with any of the two tumor types, and an oral slope factor of 0.13 per (mg/kg-day) was calculated (see Table 4.2.1 below for the tumor incidence and estimated human equivalent doses). Animal doses were adjusted for gavage dosing, and human equivalent doses were derived using a surface area correction factor in which the daily adjusted animal dose is scaled to human equivalent doses in proportion to body weight raised to the 2/3 power.

The animals that died prematurely or before the first tumor appeared at week 50 or earlier are not considered to be at risk and are not included in the total tumors used for calculation of oral unit risk.

Table 4.2.1: Dose-Response Data for the NTP Gavage Study (1985)

DOSE		TUMOR INCIDENCE			
Administered (mg/kg/day)	Human Equivalent $(BW)^{2/3}$	Urinary Bladder Carcinomas	Lung Tumors	Fore Stomach Tumors	Tumors Combined
Female mice (B6C3F1)					
0	0	0/50	2/50	0/50	2/50
21.4	1.61	8/50	4/50	1/50	12/50
42.8	3.23	21/47	8/47	4/47	28/47

DOSE		TUMOR INCIDENCE		
Administered (mg/kg/day)	Human Equivalent $(BW)^{2/3}$	Liver Tumors (Neoplastic nodules or carcinomas)	Stomach Tumors (Papillomas or Carcinomas)	Tumors Combined
Male rats (F344)				
0	0	1/49	1/49	2/49
10.7	1.94	6/47	1/47	7/47
21.4	3.89	8/50	13/50	19/50

4.2.5 Cancer Risk Evaluation Using the New Proposed Methodology

The evaluation of 1,3-DCP is described here, consistent with the principles of the proposed cancer guidelines (USEPA, 1996a) described in the FR notice (USEPA, 1998a) and in the TSD (USEPA, 1998b).

Based on sufficient evidence from animal studies (multiple tumor types in several species by oral, inhalation and dermal routes), positive mutagenicity, and structural analogues, 1,3-DCP is considered **"likely to be carcinogenic to humans by all routes of exposure."** Based on the mutagenic mode of action, a linear low dose approach is recommended.

4.2.5.1 Rationale for Selecting the Cancer Assessment Approach

In the new scheme of cancer classification, 1,3-DCP is considered as a likely human carcinogen by all routes of exposure. The classification is based on a weight of evidence approach to: (a) findings of carcinogenicity in multiple studies of rats and mice by oral, inhalation, and dermal exposures; (b) numerous positive mutagenicity assays; and (c) membership in a class of DNA-reactive compounds that cause cancer in animals and humans, including vinyl chloride, a known human carcinogen. See the proposed cancer guidelines (USEPA, 1996a) for additional information on the classification scheme.

Based on the mutagenic mode of action and the lack of information that would support a nonlinear approach to this chemical, a linear low dose approach is used.

4.2.5.2 Calculation of the Cancer Potency Factor Using the New Linear Method

A revised cancer potency value for oral exposure to 1,3-DCP was calculated by following the steps outlined in the FR notice (EPA, 1998a) and the related TSD (USEPA, 1998b). The same tumor data (urinary bladder carcinomas, lung tumors and fore stomach tumors in female mice, and liver and stomach tumors in male rats) from NTP (1985) are used as the basis for calculating the cancer potency value. The following calculations were carried out:

1) The multistage model was used to separately model the dose-response data in the observed range for female mice and male rats. Human equivalent doses were derived by adjusting animal dose in proportion to body weight raised to the 3/4 power (USEPA, 1996a). After this adjustment, the LED_{10}[3] (the lower 95th percent confidence limit on the dose at which 10 percent of the animals above background respond) was identified at 0.865 mg/kg-day using tumor data from female mice and 1.28 mg/kg-day using tumor data from male rats (see Table 4.2.2 below):

[3]Use of the LED_{10} as the point of departure is recommended with this methodology, as it is with the Proposed Cancer Guidelines. Public comments were requested on the use of the LED_{10}, ED_{10}, or other points. EPA is currently evaluating these comments, and any changes in the Cancer Guidelines will be reflected in the final AWQC methodology.

Table 4.2.2: Summary of Risk Estimates by the *new Linear Method* (NTP, 1985)*

DOSE		TUMOR INCIDENCE			
Administered** (mg/kg/day)	Human Equivalent $(BW)^{3/4}$	Urinary Bladder Carcinomas	Lung Tumors	Fore Stomach Tumors	Tumors Combined
Female mice (B6C3F1)					
0	0	0/50	2/50	0/50	2/50
21.4	3.1	8/50	4/50	1/50	12/50
42.8	6.2	21/47	8/47	4/47	28/47

DOSE		TUMOR INCIDENCE		
Administered** (mg/kg/day)	Human Equivalent $(BW)^{3/4}$	Liver Tumors (Neoplastic nodules or carcinomas)	Stomach Tumors (Papillomas or Carcinomas)	Tumors Combined
Male rats (F344)				
0	0	1/49	1/49	2/49
10.7	2.98	6/47	1/47	7/47
21.4	5.96	8/50	13/50	19/50

* -Same tumor incidence as in Table 4.2.1 except using the new scaling factor of $(BW)^{3/4}$
** -Administered by gavage in corn oil

2) linear extrapolation was carried out from response at the LED_{10} to the origin (zero dose, zero response). The slope of this line was obtained using the following equation:

$$m = \frac{0.10}{LED_{10}}$$

(Equation 4.2.1)

The variable "m" is the cancer potency value and was calculated to be 0.116 $(mg/kg\text{-}day)^{-1}$ from female mice data, and 0.078 $(mg/kg\text{-}day)^{-1}$ from male rat data. The geometric mean of these two cancer potency factors is 0.095 $(mg/kg\text{-}day)^{-1}$:

Female mice: LED_{10} =0.865 mg/kg-day

Slope = 0.116 per (mg/kg-day)

Male Rats: LED_{10} = 0.1.28 mg/kg-day
Slope = 0.078 per (mg/kg-day)

Geometric Mean of the *above* two slopes = 0.095 per (mg/kg-day) or 9.5E-2 per (mg/kg-day)

3) The risk-specific dose (RSD) was calculated for the specific incremental targeted lifetime cancer risk of one in one million (10^{-6}), one in one hundred thousand (10^{-5}), or one in ten thousand (10^{-4}) using the equation:

$$RSD = \frac{Target\ \ Incremental\ \ Cancer\ \ Risk}{m}$$

(Equation 4.2.2)

where:

RSD	=	risk specific dose (mg/kg/day)
Target Risk	=	10^{-6}
m	=	cancer potency factor of 0.095 $(mg/kg\text{-}day)^{-1}$

Thus, the calculated RSD is 1.0×10^{-5} mg/kg-day for a lifetime cancer risk of 10^{-6}. This RSD (1.0×10^{-5} mg/kg-day) is substituted into Equation 7.1.1. in Section 7.1. For a lifetime risk of 10^{-6}, the AWQC is calculated as 0.34 µg/L or 14 µg/L for ingestion of drinking water and aquatic organisms, or ingestion of aquatic organisms alone (including incidental water ingestion from recreational activities), respectively.

4.2.6 Discussion of Confidence

The carcinogenicity assessment is based on the observation of multiple tumors in two animal species exposed to the chemical by two routes. Appropriate numbers of animals were treated and observed for their lifetime in two good-quality studies with adequate dosing for carcinogenicity testing. However, only two dose levels were tested, and the chemical was administered via gavage in corn oil, not in drinking water. 1,3-DCP induced urinary bladder carcinoma, alveolar/bronchiolar adenoma, and forestomach papilloma/carcinoma in female mice at the high dose tested (HDT); and combined liver neoplastic nodules and carcinoma, and forestomach papilloma/carcinoma in male rats at the HDT. All three tumor types in female mice and the two tumor types in male rats are considered related to the administration of 1,3-DCP. No increased mortality occurred in the treated rats. In the female mice, the survival rate of the treated groups are also high (72% and 90% for the low and high-dose group, respectively, compared to 92% in the controls).

5. EXPOSURE ASSUMPTIONS

5.1 RSC Analysis

When an ambient water quality criterion is set based on non-carcinogenic effects, or carcinogenic effects evaluated by the margin of exposure (MOE) approach, anticipated exposures from non-occupational sources (e.g., food, air) are taken into account. The amount of exposure attributed to each source compared to total exposure is called the relative source contribution (RSC) for that source. The allowable dose (typically, the RfD) is then allocated via the RSC approach to ensure that the criterion is protective enough, given the other anticipated sources of exposure. Thus, accounting for non-water exposure sources results in a more stringent ambient water quality criterion than if these sources were not considered. The method of accounting for non-water exposure sources is described in more detail in the FR notice (USEPA, 1998a) and in the TSD (USEPA, 1998b). Available information on exposure sources is discussed below. However, because the criterion is based on the linear approach used to assess carcinogenicity, the information is not used to determine an RSC for 1,3-DCP.

5.1.1 Overview of Potential for Exposure

Throughout this Section, the studies refer to both 1,3-DCP and just DCP. Where the studies specify the isomer, it has been included as such. Where the studies have not specified, it is referred to as DCP.

DCP is used as a soil fumigant to control nematodes on crops grown in sandy soils of most of the United States. Since the pesticide use of ethylene dibromide and dibromochloropropane have been canceled, the use of DCP has increased.

The National Agricultural Statistics Service's (NASS) Field Crops Summary is an annual report of on-farm use of agricultural chemicals. In the most recent Field Crops Summary, use of fertilizers and pesticides on corn, wheat, cotton, peanut, potatoes, rice, sorghum, and soybeans are reported for United States farms in 1991. According to the report, DCP has been applied to crops in Idaho, Oregon, and Washington. Results are shown in Table 5.1.1 (NASS, 1991).

Table 5.1.1: NASS Agricultural Use Data for 1,3-Dichloropropene

Crop	State	Total Applied (1,000 lbs)
Fall potatoes	Idaho	1,810
Fall potatoes	Oregon	1,374
Fall potatoes	Washington	5,247

According to the EPA's Toxics Release Inventory, the total release of DCP into the environment in 1990 by manufacturers was 65,734 pounds. The two largest pathways of release were emissions to air, accounting for 98% (64,379 pounds), and releases to water (accounting for less than 2% or 1,025 pounds). Underground injection was reported at 330 pounds and there was no reported release onto land (USEPA, 1995a).

EPA's National Toxics Inventory data base reported air emissions of 18,820,000 pounds/year (USEPA, 1996b). The value from the National Toxics Inventory is much higher than the reported releases from the Toxics Release Inventory, in part because the National Toxics Inventory data base includes emissions from combustion of coal, which is not reported to the Toxics Release Inventory (French, 1996). The large disparity may also be due to additional factors. However, information to describe this difference is not available.

5.1.2 Occurrence in Environmental Media

The following sections describe studies that measured concentrations of 1,3-DCP in environmental media.

5.1.2.1 Exposure from Drinking Water Systems and Source Water

Two studies that have analyzed drinking water for DCP are discussed here. As part of the EPA's National Survey of Pesticides in Drinking Water Wells (National Pesticide Survey), 566 community water system (CWS) wells and 783 rural domestic drinking water wells were surveyed for 127 pesticides, pesticide degradates, and nitrate from 1988 to 1990. In this survey, the cis- and trans- isomers of DCP were each analyzed for. Neither cis- or trans-DCP were detected at or above the minimum reporting limits (MRL) of 0.010 and 0.10 µg/L, respectively. Based on the precision of the survey, EPA estimates that the maximum number of wells that may contain either cis- or trans-DCP is 750 (0.8%) CWS wells and 83,100 (0.8%) rural domestic wells based on a 95% upper-bound confidence level (USEPA, 1990).

In the other survey, from September 1981 to January 1982, the New York State Department of Health conducted a study of organic chemical contamination of selected community water systems in the state. Eighty drinking water samples were evaluated from 69 community water systems, of which 59 had ground-water supplies, nine surface water supplies, and three mixed-water supplies. There were no detections of either cis- or trans-DCP. The detection limit was 1 µg/L for both (Close et al., 1982).

The EPA's Unregulated Contaminant Data Base was searched for occurrence data for DCP in drinking water. A total of 25 states reportedly monitored for DCP with nine states reporting positive results. Results from each state that detected DCP are shown in Table 5.1.2.

Table 5.1.2: Unregulated Contaminant Data Base Results for 1,3-Dichloropropene

State	Source	Number of Facilities	Number of Positives	Maximum (µg/L)
Alabama	Ground	162	1	1.6
Massachusetts	Ground	4	3	17.0
Maryland	Ground	132	1	0.3
Missouri	Ground	265	1	0.2
Ohio	Ground	5,896	2	1.0
Delaware	Surface	83	1	1.2
Massachusetts	Surface	3	3	1.2
Pennsylvania	Surface	230	6	1.55
Texas	Unknown	2	2	12.0

In addition to the drinking water surveys, several studies examined DCP in source water, seven of which surveyed ground water. In California, 54 wells in areas of extended DCP use were sampled. No positive detections were found based on a detection limit of 0.1 µg/L (Maddy et al., 1982, as cited in USEPA, 1988).

Parsons and Witt (1989) surveyed all 50 state lead agencies for information regarding pesticides in ground water of their respective states. Five states reported sampling of DCP, of which four reported detections of DCP in ground water. From a total of 5,517 wells sampled, 5,510 wells did not contain detectable levels, two had levels less than or equal to 2.2 µg/L, and 5 wells were >2.2 µg/L.

The Pesticides in Ground Water Data Base is a compilation of monitoring data for pesticides in ground water originating from studies conducted by pesticide registrants, universities, and government agencies. Additional data was obtained from published literature and direct correspondence with sponsors of the studies. For DCP, compiled studies covered seven states and 21,072 wells. Results are shown in Table 5.1.3 (USEPA, 1992b).

Table 5.1.3: Pesticides in Ground Water Data Base Results for 1,3-Dichloropropene				
State	**Sample Year(s)**	**Number of Wells Sampled**	**Number of Wells Detected**	**Range (µg/L)**
California	1979-1989	5,364	3	0.890-31
Florida	1909-1991	15,281	2	0.28-7.8
Hawaii	1979-1987	54	0	-
Massachusetts	1985	239	0	-
Missouri	1989-1990	198	0	-
New York	1983-1985	17	1	18-140
Oregon	1985-1987	117	0	-

In 1987, effects of land use on ground-water quality in central Florida was studied by the U.S. Geological Survey in cooperation with the Florida Department of Environmental Regulation. Ground water was sampled in four areas of different land uses: an urban area, a citrus farming area, a phosphate mining area, and a control area. Out of 32 samples from the four areas, no measurable amounts of DCP were found. The detection limit was 3.0 µg/L (Rutledge, 1987).

In 1986, the Rhode Island Department of Environmental Management conducted a state-wide survey of private wells (also known as the Private Well Survey), which collected 485 samples from 458 wells and analyzed them for a variety of contaminants, including DCP. Wells were chosen from areas where land use presented a potential threat to ground water, with additional background wells chosen from relatively uncontaminated areas. Neither cis- nor trans-DCP was detected in any of the samples analyzed. Detection limits were not reported (RIDEM, 1990).

The Well Inventory Data Base, developed by the California Department of Food and Agriculture (CDFA) in 1983, is a state-wide data base containing pesticide sampling results from untreated unfiltered California wells. In 1990, the data base was updated with results submitted to the CDFA from July 1989 to June 1990 and included 28 studies conducted between 1987 and 1989. A total of 1,482 observations from 1,307 wells were reported for DCP, with two unconfirmed detections. Unconfirmed results are defined as single detections which were not confirmed by subsequent analyses (Miller et al., 1990).

Information is available on 1,3-DCP in ambient surface waters. Hall et al. (1987) sampled the Potomac River at Quantico, Virginia for several contaminants. Based on a detection limit of 2 µg/L, DCP was not detected. Merriman et al. (1991) found 1,3-DCP in surface waters of an Ontario watershed situated in prime agricultural land. Combined concentrations of the cis-and trans-isomers in the detected samples ranged from 0.18 µg/L to 4.12 µg/L. Less than 10% of the samples had detectable levels of 1,3-DCP.

STORET, operated by the EPA, is a computerized data base comprising water quality data collected from states, EPA Regional offices, and other government agencies. It contains over 130 million observations for over 700,000 sampling sites located throughout the United States. It is important to note that there are limitations in using STORET data to estimate representative concentrations of contaminants in public water systems. The data in STORET were collected from an array of studies conducted for various purposes. Analyses were conducted in different laboratories employing different methodologies with a range of detection limits. In many cases the detection limits were not reported. In ambient water, there was one positive detection of DCP in Utah. A concentration of 1.9 µg/L was reported (USEPA, 1992c).

5.1.2.2 Dietary and Fish Exposures

The Food and Drug Administration, as part of their enforcement of pesticide tolerances, analyzed 13,085 food samples in 1989 and 15,000 in 1987 for nearly 250 pesticides. Food samples were collected from locations and during harvest periods most likely to produce samples with pesticide residues. DCP was not detected in any of the samples analyzed and the detection limit was not reported (USFDA, 1988; USFDA, 1990).

A search of the literature found no information on the presence of 1,3-DCP in fish, and 1,3-DCP was not included in the National Study of Chemical Residues in Fish (USEPA, 1992d).

5.1.2.3 Respiratory Exposures

Shah and Heyerdahl (1988) reviewed published literature and unpublished data, compiling ambient air monitoring data from 1970 to 1987. A total of 148 urban air samples were reported for DCP, with a mean of 116 $\mu g/m^3$ (23.4 ppb) and a median of 118 $\mu g/m^3$ (23.9 ppb). The lower and upper quartiles were 37 $\mu g/m^3$ (7.5 ppb) and 176 $\mu g/m^3$ (35.5 ppb). No indoor concentrations were reported (McAllister et al., 1986, as cited in Shah and Heyerdahl, 1988).

Pellizzari et al. (1979) conducted a study to survey the occurrence of halogenated hydrocarbons in various environmental media in five metropolitan areas. As part of this study, Pellizzari et al. (1979) combined DCP concentrations in ambient air at one site with additional data from other research programs. In the Baton Rouge, Louisiana area, two of 11 samples (18%) were positive, with concentrations ranging from trace amounts to 10 ng/m^3. This survey is included in Shah and Heyerdahl (1988) above.

Four studies monitored for 1,3-DCP in ambient air in urban areas of the U.S. during the late 1980s and early 1990s (USEPA, 1994a). The four studies included a variety of cities throughout the U.S. No positive concentrations of the pollutant were detected, and the detection limit was not given.

5.2 Exposure Data Adequacy and Estimate Uncertainties

Numerous studies sampled for DCP in public drinking water supplies, drinking water wells, and in ambient waters. Of the two national studies, DCP was not found above detection limits in one and occurred infrequently in the other. In the various compilation studies or individual State studies, DCP was found infrequently. These infrequent detections include both drinking waters and ambient waters, and represent generally low concentrations. Based on the available data, it appears that DCP is not a commonly occurring contaminant at significant levels in drinking water supplies or ambient waters. The amount of information on DCP in dietary foods is very limited. However, FDA samplings from 1987 and 1989 indicated no detectable amounts of DCP in the samples analyzed. No information is available to characterize the potential exposure from fish consumption. More information is needed to adequately assess the potential for exposure to acrylonitrile from the diet and especially from fish. Additionally, the amount of data on concentrations in ambient air is limited. Two available compilation studies indicate the potential for exposure from air in urban/metropolitan locations. These studies could represent levels that persons living in urban areas may be exposed to. However, more information is necessary to adequately assess the likelihood and potential range of exposure to DCP from ambient air, especially given the large amount of emissions into the air.

The exposure parameters used for national criteria for 1,3-DCP reflect exposures for the general adult population. These exposure parameters are chosen for several reasons. First, sufficient information on the toxicological susceptibility of specific populations (specifically, pregnant women and children) is not available for 1,3-DCP. In addition, it is not clear whether a particular population is likely to be more highly exposed than another population from common sources. Farm workers may be more highly exposed via pesticide application activities or consumption of water from wells or other supply sources at the farm. Such occupational exposures are not included in this analysis. However, it is acknowledged that these workers are likely to experience 1,3-DCP exposures greater than the general population. Although infants and children have a higher rate of water and food consumption per body weight compared to adults (USEPA, 1994b), the cancer estimates are based on lifetime exposures. Therefore, the criterion for 1,3-DCP is evaluated using exposure factors applicable to adults. Also, although certain water bodies may support populations of sport fishers and subsistence fishers who eat more fish than the general population, these national criteria are derived to protect the majority of the general population.

5.3 Exposure Intake Parameters

Exposure parameters (e.g., fish intake, drinking water intake, and body weight) used in the Ambient Water Quality Criteria equation should reflect the population to be protected. Default exposure parameters are available for the general population of adults as well as several specific populations that may be highly exposed or more toxicologically susceptible to a given chemical (USEPA, 1998a and 1998b).

The exposure parameters and values for the general population of adults are as follows:

Fish intake (FI)	0.01780 kg/day
Drinking water intake (DI)	2 L/day (used for drinking water sources)
Incidental ingestion (II)	0.01 L/day (used for non-drinking water sources)
Body Weight (BW)	70 kg

6. BIOACCUMULATION FACTOR

This section describes the procedures and data sources used to calculate the bioaccumulation factor (BAF) used for deriving an ambient water quality criterion for 1,3-dichloropropene. Details and the scientific basis of EPA's recommended methodology for deriving BAFs are described in USEPA (1998a) and in USEPA (1998b). When determining a BAF for use in deriving ambient water quality criteria (AWQC) for nonpolar organic chemicals, two general steps are required. The first step consists of calculating baseline BAFs for organisms at appropriate trophic levels using available field and laboratory studies of the bioaccumulation or bioconcentration of the chemical of interest. Since baseline BAFs are normalized by important factors shown to affect bioaccumulation (e.g., the lipid content of aquatic organisms on which they are based, the freely dissolved concentration of the chemical in water), they are more universally applied than BAFs not adjusted for these factors. Once baseline BAFs have been calculated for the appropriate trophic levels, the second step involves adjusting the baseline BAFs to reflect the expected conditions at the sites that are applicable to the AWQC (e.g., lipid content of consumed organisms and the freely dissolved fraction of the chemical in the site water). Application of both of these steps to the derivation of a BAF for 1,3-dichloropropene is described below.

6.1 Baseline BAF

Different procedures are recommended by EPA for determining the baseline BAF depending on the type of bioaccumulation data available. As described in USEPA (1998b), the data preference for deriving a BAF for nonpolar organics is (in order of preference):

1. Calculation of a baseline BAF from a reliable field-measured BAF,
2. Calculation of a baseline BAF from a reliable field-measured biota-sediment accumulation factor BSAF,
3. Calculation of a baseline BAF from a laboratory-measured (bioconcentration factor (BCF) and food-chain multiplier (FCM), and
4. Calculation of a baseline BAF from a predicted BCF and FCM.

For 1,3-DCP, no acceptable measured BAF, BSAF, or BCF was found. Therefore, method 4 above was chosen for determining the baseline BAF. This method is described further in USEPA (1998b). According to this method, the baseline BAF is determined for each trophic level as:

$$\text{Baseline BAF}_{\ell}^{fd} = (\text{BCF}_{\ell}^{fd}) \cdot (\text{FCM}) = (K_{ow}) \cdot (\text{FCM})$$

(Equation 6.1.1)

where:

Baseline BAF_{ℓ}^{fd}	=	predicted baseline BAF (L/kg-lipid) that, if measured, would reflect the lipid-normalized concentration in the biota divided by the freely dissolved concentration in the water for aquatic organisms occupying a designated tropic level,
BCF_{ℓ}^{fd}	=	baseline BCF expressed on a freely-dissolved and lipid-normalized basis,
FCM	=	food-chain multiplier reflecting biomagnification at the designated trophic level (unitless), and
K_{ow}	=	octanol-water partition coefficient.

Fish consumption rates determined from the USDA's Continuing Survey of Food Intakes by Individuals (CSFII) indicate that on a national, average per capita basis, individuals in the United States consume significant quantities of fish and shellfish at trophic levels two (e.g., clams, oysters), three (e.g., crab, shrimp, flounder) and four (e.g., trout, pike, certain catfish species) (USEPA, 1998c). Therefore, the national AWQC for 1,3-DCP requires that baseline BAFs be derived to reflect bioaccumulation in aquatic organisms at each of these three trophic levels.

For 1,3-DCP, a baseline BAF of 57.5 was calculated for organisms at trophic levels two, three and four using equation 6.1.1. These calculations are shown below for each trophic level.

<u>Trophic Level Two</u>:

Baseline $\text{BAF}_{\ell}^{fd} = (K_{ow})\bullet(\text{FCM2})$
$= (10^{1.76})\bullet(1.000)$
= 57.5 L/kg-lipid (expressed as 3 significant digits for convenience)

<u>Trophic Level Three</u>:

Baseline $\text{BAF}_{\ell}^{fd} = (K_{ow})\bullet(\text{FCM3})$
$= (10^{1.76})\bullet(1.000)$
= 57.5 L/kg-lipid (expressed as 3 significant digits for convenience)

<u>Trophic Level Four</u>:

Baseline $\text{BAF}_{\ell}^{fd} = (K_{ow})\bullet(\text{FCM4})$
$= (10^{1.76})\bullet(1.000)$
= 57.5 L/kg-lipid (expressed as 3 significant digits for convenience)

The calculated baseline BAFs do not differ at each trophic level because the relatively low K_{ow} of 1,3-DCP (57.5 or $\log_{10} K_{ow}$ of 1.76) results in predicted FCMs of 1.000 at each trophic level. A log K_{ow} of 1.76 was selected as a typical value based on the predicted log K_{ow} from the CLOGP program by Hansch and Leo (1995). Values of 1.000 were selected as the FCMs at trophic levels two, three, and four according to FCMs recommended in the Technical Support Document (TSD) for the AWQC Methodology Revisions for organic chemicals with a log K_{ow} of 2.0 or less (USEPA, 1998b).

6.2 AWQC BAF

After the derivation of trophic level-specific baseline BAFs for 1,3-DCP (described in the previous section), the next step is to calculate BAFs that will be used in the derivation of AWQC. This step is necessary to adjust the baseline BAFs to conditions that are expected to affect the bioavailability of 1,3-DCP at the sites applicable to the AWQC. Derivation of the AWQC BAF requires information on: (1) the baseline BAF at appropriate trophic levels, (2) the percent lipid of the aquatic organisms consumed by humans at the site(s) of interest (trophic level specific), and (3) the freely dissolved fraction of the chemical in ambient water at the site(s) of interest. For each trophic level, the equation for deriving a BAF to used in deriving AWQC is:

$$\text{BAF for AWQC}_{(\text{TL } n)} = [(\text{Baseline BAF}_{\ell}^{fd})_{\text{TL } n} \cdot (f_{\ell})_{\text{TL } n} + 1] \cdot (f_{fd})$$

(Equation 6.2.1)

where:

BAF for AWQC $_{(\text{TL n})}$	=	BAF at trophic level “n” used to derive AWQC based on site conditions for lipid content of consumed aquatic organisms for trophic level “n” and the freely dissolved fraction in the site water
Baseline BAF_{ℓ}^{fd} $_{(\text{TL n})}$	=	BAF expressed on a freely dissolved and lipid-normalized basis for trophic level “n”
$f_{\ell\ (\text{TL n})}$	=	Fraction lipid of aquatic species consumed at trophic level “n”
f_{fd}	=	Fraction of the total chemical in water that is freely dissolved

Each of the equation components is discussed below.

6.2.1 Baseline BAFs (Baseline BAF_{ℓ}^{fd})

The derivation of baseline BAFs at specific trophic levels is described in Section 6.1. For 1,3-DCP, a baseline BAF of 57.5 was derived for aquatic organisms at trophic levels two, three and four.

6.2.2 Lipid Content of Consumed Aquatic Species (f_ℓ)

Accumulation of nonpolar organic chemicals in aquatic organisms has repeatedly been shown to be a function of lipid content (e.g., Mackay, 1982; Connolly and Pedersen, 1988; Thomann, 1989). Therefore, baseline BAFs, which are lipid normalized for comparative purposes, need to be adjusted to reflect the lipid content of aquatic organisms consumed by the target population. As discussed in the FR Notice and TSD (USEPA, 1998a,b), EPA recommends that where possible, lipid content of consumed aquatic species be determined on a consumption-weighted average basis.

For the purposes of deriving national ambient water quality criteria, EPA has established national default, consumption-weighted lipid content values of 2.3% at trophic level two, 1.5% at trophic level three, and 3.1% at trophic level four. These national default lipid content values are based on a national survey of fish and shellfish consumption rates and information on their lipid content (see USEPA 1998a, 1998b for details of the determination of national default lipid content values). As discussed in the FR Notice and TSD (USEPA, 1998a, 1998b), EPA considers the use of national default lipid values as being appropriate in situations where local or regional data on lipid content and consumption rates are unavailable for the site(s) applicable to the AWQC. However, if local or regional data are available for the site(s) of interest, EPA recommends that States and Tribes use the local or regional data instead of the national default values because the type and quantity of consumed aquatic organisms and their lipid content may vary from one location to another.

6.2.3 Freely-Dissolved Fraction Applicable to AWQC

Information on the freely-dissolved fraction of the chemical expect at the site(s) applicable to the AWQC is important because the freely dissolved form of nonpolar organic chemicals is considered to represent the most bioavailable form in water and thus, the form that best predicts bioaccumulation (USEPA 1998a, 1998b). Freely dissolved chemical is defined as the portion of the chemical dissolved in water, excluding the portion sorbed on to particulate and dissolved organic carbon. The freely-dissolved fraction is estimated from the octanol-water partition coefficient and the dissolved and particulate organic carbon concentrations as shown below.

$$f_{fd} = \frac{1}{\left[1 + (POC \cdot K_{ow}) + (DOC \cdot \frac{K_{ow}}{10})\right]}$$

(Equation 6.2.2)

where:

f_{fd} = freely-dissolved fraction of chemical in water applicable to the AWQC
POC = concentration of particulate organic carbon applicable to the AWQC (kg/L)

DOC	=	concentration of dissolved organic carbon applicable to the AWQC (kg/L)
K_{ow}	=	n-octanol water partition coefficient for the chemical

In this equation, the terms “K_{ow}” and “K_{ow}/10” are used to estimate the partition coefficients to POC and DOC, respectively, which have units of L/kg, the scientific basis of which is explained in USEPA (1998b). Based on national default values of 2.9 mg/L for DOC, 0.48 mg/L for POC, and 57.5 for the K_{ow} (Log K_{ow} of 1.76), the freely dissolved concentration of 1,3-DCP is calculated to be 1.000 (expressed as four significant digits for convenience). Calculation of the default freely dissolved concentration is provided below.

$$f_{fd} = \frac{1}{[1 + (POC \cdot K_{ow}) + (DOC \cdot \frac{K_{ow}}{10})]}$$

$$f_{fd} = \frac{1}{[1 + (4.8 \text{ x } 10^{7} \text{ kg/L} \cdot 57.5 \text{ L/kg}) + (2.9 \text{ x } 10^{6} \text{ kg/L} \cdot \frac{57.5}{10} \text{ L/kg})]}$$

$$f_{fd} = 1.000$$

The national default values for POC and DOC used here are based on the median value of POC and DOC concentrations observed in numerous water bodies across the United States and are described further in USEPA (1998a,b). For the purposes of deriving national AWQC, EPA believes that the use of national default values is appropriate. In addition, EPA considers the use of national default values of POC and DOC as being appropriate in situations where local or regional data on POC and DOC are unavailable for the site(s) applicable to the AWQC. However, if local or regional data are available for the site(s) of interest, EPA recommends that States and Tribes use the local or regional data instead of the national default values because the POC and DOC can vary on a local basis, thus affecting the freely dissolved fraction.

6.2.4 Calculation of AWQC BAFs

Using equation 6.2.1 above, BAFs appropriate for calculating national AWQC are: 2.32, 1.86, and 2.78 for organisms at trophic levels two, three and four, respectively (expressed as three significant digits for convenience). These BAFs were derived using 57.5 L/kg for the baseline BAF at all three trophic levels, percent lipid content values of 2.3%, 1.5%, and 3.1% at trophic levels two, three, and four, respectively, and a freely dissolved fraction of 1.000. Calculation of the AWQC BAFs are shown below.

$$\text{BAF for AWQC}_{(\text{TL } n)} = [(\text{Baseline BAF}_{\ell}^{fd})_{\text{TL } n} \cdot (f_{\ell})_{\text{TL } n} + 1] \cdot (f_{fd})$$

AWQC BAF for Trophic Level Two

= [(57.5 L/kg-lipid)•(0.023) +1] • (1.000)

= 2.32 L/kg-tissue (expressed as three significant digits for convenience)

AWQC BAF for Trophic Level Three

= [(57.5 L/kg-lipid)•(0.015) +1] • (1.000)

= 1.86 L/kg-tissue (expressed as three significant digits for convenience)

AWQC BAF for Trophic Level Four

= [(57.5 L/kg-lipid)•(0.031) +1] • (1.000)

= 2.78 L/kg-tissue (expressed as three significant digits for convenience)

7. AWQC CALCULATION

7.1 For Ambient Waters Used as Drinking Water Sources

The cancer-based AWQC was calculated using the RSD and other input parameters listed below:

$$\text{AWQC} = \text{RSD x} \left(\frac{\text{BW}}{\text{DI} + \sum_{i=2}^{4} (\text{FI}_i \text{ x } \text{BAF}_i)} \right)$$

(Equation 7.1.1)

where:

RSD	=	Risk specific dose 1.0 x 10^{-5} mg/kg-day (10^{-6} Risk)
BW	=	Human body weight assumed to be 70 kg
DI	=	Drinking water intake assumed to be 2 L/day
FI_i	=	Fish intake at trophic level i, i=2,3, and 4;total intake assumed to be 0.01780 kg/day[4]
BAF_i	=	Bioaccumulation factor at trophic level i (i=2, 3, and 4) equal to 2.32 L/kg-tissue for trophic level two, 1.86 L/kg-tissue for trophic level three, and 2.78 L/kg-tissue for trophic level four

[4] Fish intake rates for each trophic level are: TL2=0.0011 kg/day; TL3=0.0115 kg/day; and TL4=0.0052 kg/day (presented as four significant figures for convenience). For further information, see Section 2.4.8 of the TSD.

This yields a value of 3.4 x 10^{-4} mg/L, or 0.34 µg/L (rounded from 0.343 µg/L).

7.2 For Ambient Waters Not Used as Drinking Water Sources

When the water body is to be used for recreational purposes and not as a source of drinking water, the drinking water value (DCR above) is eliminated from the equation and it is substituted with an incidental ingestion value (II). The incidental intake is assumed to occur from swimming and other activities. The fish intake value is assumed to remain the same. The default value for incidental ingestion is 0.01 L/day. When the above equation is used to calculate the AWQC with the substitution of an incidental ingestion of 0.01 L/day, an AWQC of 1.4 x10^{-2} mg/L, or 14 µg/L (rounded from 14.46 µg/L) is obtained.

8. SITE-SPECIFIC OR REGIONAL ADJUSTMENTS TO CRITERIA

Several parameters in the AWQC equation can be adjusted on a site-specific or regional basis to reflect regional or local conditions and/or specific populations of concern. These include fish consumption; incidental water consumption as related to regional/local recreational activities; BAFs (including factors used to derive BAFs such as POC/DOC, percent lipid of fish consumed by target population, and species representative of given trophic levels); and the relative source contribution. States and Tribes are encouraged to make adjustments using the information and instructions provided in the TSD (USEPA, 1998b).

9. REFERENCES

Clayton, G.D. and F.E. Clayton. 1981. Patty's Industrial Hygiene and Toxicology. 3rd Edition Volume 2B. New York, New York. J. Wiley & Sons Ltd. Pp. 3573-3577

Close, J., K. Slade, and K. Markussen. 1982. Report of 1981 Organic Chemical Surveillance Survey at Community Water Systems in New York State. New York State Department of Health. August.

Climie, I.J., D.H. Hutson, B.D. Morrison and G. Stoydin. 1979. Glutathione conjugation in the detoxication of (Z)-1,3-dichloropropene (a component of nematocide D-D) in the rat. Xenobiotica. 9(3):149-156.

Connolly, H. and C. Pedersen. 1988. A thermodynamic-based Evaluation of Organic Chemical Accumulation in Aquatic Organisms. Environ. Sci. Technol. 22: 99-103.

Creedy, C.L., T.M. Brooks, B.J. Dean, D.H. Hutson and A.B. Wright. 1984. The protective action of glutathione on the E-isomers of 1,3-dichloropropene. Chem. Biol. Interact. 50(1): 39-48.

De Lorenzo, F., S. Degl'Innocenti, A. Ruocco, L. Silengo and R. Cortese. 1977. Mutagenicity of pesticides containing 1,3-dichloropropene. Cancer Res. 37(6): 1915-1917.

Dietz, F.K., E.A. Hermann, P.E. Kastl, D.A. Dittenber and J.C. Ramsey. 1985. 1,3-Dichloropropene: Pharmacokinetics, effects on tissue non-protein sulfhydryls and marcomolecular binding in Fischer 344 rats and B6C3F1 mice following oral administration. Unpublished study prepared and submitted by Dow Chemical USA, Midland, MI under TSCA 8d. OTS50515836.

Fisher, G.D. 1988. The disposition of 1,3-dichloropropene in the rat following acute inhalation exposure. Diss. Abstr.Int.B. 48(12 Pt 1):3537.

Fisher, G.D. and W.W. Kigore. 1988. Tissue levels of glutathione following acute inhalation of 1,3-dichloropropene. J. Toxicol. Environ. Health. 23(2): 171-182

French, C. 1996. USEPA. Personal communication with Amy Benson, Abt Associates, Bethesda, MD. August 2.

Gosselin, R., H. Hodge, R. Smith and M. Gleason III. 1976. Dichloropropenes. Clinical Toxicology of Commercial Products, 4th ed. Williams and Wilkins, Baltimore, MD. P. 119-121.

Hall, L.W., Hall, W.S., Bushong, S.J., and R. L. Herman. 1987. *In situ* striped bass (*Morone saxatilis*) contaminant and water quality studies in the Potomac River. Aquatic Toxicology 10:73-99.

Hansch, C. and A. Leo. 1995. Exploring QSAR. American Chemical Society.

Haworth, S., T. Lawlor, K. Mortelmans, W. Speck and E. Zieger. 1983. Salmonella mutagenicity test results for 250 chemicals. Environ. Mutagen. 5(Suppl. 1): 3-142.

Hine, C.H, H.H.Anderson, H.D. Moon, J.K. Kodama, M. Morse and N.W. Jacobsen. 1953. Toxicology and safe handling of CBP-55 (technical 1-chloro-3-bromopropene-1). Am. Med. Assoc. Arch. Ind. Hyg. Occup. Med. 7:118-136. (Cited in USEPA, 1980)

Hutson, D.J., J.A. Moss and Pickering. 1971. The excretion and retention of components of the soil fumigant D-D and their metabolites in the rat. Food Cosmet. Toxicol. 9:677-680.

IARC (International Agency for Research on Cancer). 1987. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Vol: 401. p. 11-130, WHO, Lyon, France.

IARC (International Agency of Research on Cancer). 1979. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Vol:19: 377, WHO, Lyon, France.

IARC (International Agency of Research on Cancer). 1974. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Vol: 7:291. WHO, Lyon, France.

IRIS, 1996 (Integrated Risk Information System), EPA. Available on line or through Toxnet, or on disk via NTIS EPA, Washington, D.C.

Jones, J.R. and T.A. Collier. 1986. Telone II: OECD 401. Acute oral toxicity test in the rat. Unpublished study prepared by Safepharm Laboratories and submitted by Dow Chemical Europe S.A., Horgen Switzerland under TSCA 8d. OST0505823.

Lomax, L.G., W. T. Stott, K.A. Johnson, LL. Calhoun, B.L. Yano and J.F. Quast. 1989. The chronic toxicity and oncogenicity of inhaled technical-grade 1,3-Dichloropropene in rats and mice. Fund. Appl. Toxicol. 12: 418:431.

Mackay, D. 1982. Correlation of Bioconcentration Factors. Environ. Sci. Technol. 16: 274-278.

Maddy, K.J.; Fong, J.L.; Conrad, D.; Fredrickson, A. 1982. A Study of Well Water in Selected California Communities for Residues of 1,3-Dichloropropene, Chloroallyl Alcohol, and 49 Organophosphate or Chlorinated Hydrocarbon Pesticides. Bull. Enviro. Contam. Toxicol., Vol. 29: 354-359. Cited in U.S. EPA, 1988.

Markovitz, A. and W.H. Crosby. 1994. Chemical carcinogenesis: A soil fumigant, 1,3-Dichloropropene, as possible cause of hematologic malignancies. Arch. Int. Med. 144(7): 1409-1411.

McAllister, R.A., Jongleux, R., Dayton, D.P., O'Hara, P, and D. Wagoner. 1986. 1986 Non-Methane Organic Compounds Monitoring; Draft Report. Radian Corporation, Research Triangle Park, NC. Prepared for the U.S. Environmental Protection Agency. EPA 68-02-3889. January. [Cited in Shah and Heyerdahl (1988)].

Merriman, J.C., J. Struger, and R.S. Szawiola. 1991. Distribution of 1,3-dichloropropene and 1,2-dichloropropane in Big Creek Watershed. Bull. Environ. Contam. Tox. 47: 572-579.

Miller, C.; Pepple, M.; Troiano, J.; Weaver, D.; Kimaru, W. 1990. Sampling for Pesticide Residues in California Well Water. State of California Department of Food and Agriculture, Environmental Hazards Assessment Program. December 1.

NASS. 1991. Field Crops Summary. National Agricultural Statistics Service funded through the Presidents's Water Quality Initiative. Data base.

NTP (National Toxicology Program). 1985. Toxicology and Carcinogenesis Studies of Telone II in F344/N Rats and B6C3F1 Mice. U.S. Dept. Health and Human Services. NTP TR 269. NIH Publ. No. 85-2525.

Neudecker, T., A. Stefani and D. Heschler. 1977. In vivo mutagenicity of soil nematocide 1,3-dichloropropene. Experientia. 33: 1084-1085.

Parsons, D.W.; Witt, J.M. 1989. Pesticides in Groundwater of the United States of America: Report of a 1988 Survey of State Lead Agencies. Conducted as Part of the National Pesticide Impact Assessment Program, Oregon State University Extensive Service, EM 8406. August.

Pellizzari, E.D., Erickson, M.D., and R.A. Zweidinger. 1979. Formulation of a preliminary assessment of halogenated organic compounds in man and environmental media. U.S. Environmental Protection Agency, Office of Toxic Substances. Washington, DC. Contract No. 68-01-4731. July. Pp. 27-83

RIDEM (Rhode Island Department of Environmental Management). 1990. Rhode Island Private Well Survey Final Report. Rhode Island Department of Environmental Management, Groundwater Section, Providence, RI. May.

Rutledge, A.T. 1987. Effects of Land Use on Ground Water Quality in Central Florida -- Preliminary Results: U.S. Geological Survey Toxic Waste -- Ground Water Contamination Program. U.S.Geological Survey, Water Resources Div., Tallahassee, FL.

Shah, J.J. and E.K. Heyerdahl. 1988. National ambient volatile organic compounds (VOCs) data base update. Nero Associates, Portland, Oregon. Prepared for Atmospheric Sciences Research Laboratory, Research Triangle Park, NC. Contract No. 68-02-4190. February.

Stolzenberg, S. and C. Hine. 1980. Mutagenicity of 2- and 3-carbon halogenated compounds in Salmonella/mammalian microsome test. Environ. Mutagen. 2: 59-66.

Stott, W.T. and P.E. Kastl. 1986. Inhalation pharmacokinetics of technical grade 1,3-dichlorpropene in rats. Toxicol. Appl. Pharmacol. 85(3): 332-341.

Sudo, S., M., Nakazawa and M. Nakazono. 1978. The mutagenicity test on 1,3-dichloropropene in bacteria test systems. Prepared by Nomura Sogo Research Institute, submitted by Dow Chemical USA, Midland, MI. MRID 39688.

Thomann, R.V. 1989. Bioaccumulation Model of Organic Chemical Distribution in Aquatic Food Chains. Environ. Sci. Technol. 23: 699-707.

Til, H.P., M.T. Spangers, V.J. Ferom, P.J. Reuzel. 1973. Sub-chronic (90 day) toxicity study with Telone in albino rats. Report Number R4002. Final Report. Unpublished Study (Central Institute for Nutrition and Food Research). Submitted by Dow Chemical, USA, Midland, MI. MRIDs 39684, 67977.

Torkelson, T. R. and V.K. Rowe. 1981. Halogenated aliphatic hydrocarbons containing chlorine, bromine and iodine. In: Patty's Industrial Hygiene and Toxicology, Vol.11B, 3rd ed., G.D. Clayton and F.E. Clayton, Ed. John Wiley and Sons, Inc. New York. p. 3573-3577.

Toyoshima, S., R. Sato, and S. Sato. 1978a. The acute toxicity test of Telone II in rats. Unpublished study prepared by Japan Experimental Research Co. And submitted by Dow Chemical USA, Midland, MI under TSCA 8d. OST0515832.

Toyoshima, S., R. Sato and S. Sato. 1978b. The acute toxicity test of Telone II in mice. Unpublished study prepared by Japan Experimental Research Co. And submitted by Dow Chemical USA, Midland, MI under TSCA 8d. OST0515833.

USEPA. 1980. Water Quality Criteria Documents; Availability. Federal Register. 45(231): 79318-79379.

USEPA. 1986. Guidelines for carcinogen risk assessment. 51 Federal Register No. 185. pp. 33992-34003.

USEPA. 1988. Health Advisories for 50 Pesticides (Including Acifluorfen, Ametryn, Ammonium Sulfamate, Atrazine, Baygon, Bentazon, Bromacil, Butylate, Carbaryl, Carboxin, Chloramben, etc.). U.S. Environmental Protection Agency, Office of Drinking Water.

USEPA. 1989. Drinking water health advisory: Pesticides. 1,3-Dichloropropene. United States Environmental Protection Agency, Office of Drinking Water Health Advisory, Lewis Publisher Inc., Chelsea, MI.

USEPA. 1990. National Pesticide Survey: Summary of Results of EPA's National Survey of Pesticides in Drinking Water Wells. U.S. Environmental Protection Agency, Office of Water and Office of Pesticides and Toxic Substances. Fall.

USEPA. 1992a. Water Quality Standards; Establishment of Numeric Criteria for Priority Toxic Pollutants; States' Compliance. Final Rule. Federal Register. 57(246): 60848-60923.

USEPA. 1992b. Pesticides in Ground Water Database: A Compilation of Monitoring Studies: 1971-1991. National Summary. USEPA, Prevention Pesticides and Toxic Substances. 734-12-92-001. September.

USEPA. 1992c. On-line search of STORET database maintained by EPA. Search completed by Wade Miller Associates, Inc., November 17.

USEPA. 1992d. National Study of Chemical Residues in Fish: Volumes I and II. Office of Science and Technology. Washington, D.C. EPA 823-R-92-008a and EPA 823-R-92-008b.

USEPA. 1994a. A Screening Analysis of Ambient Monitoring Data for the Urban Area Source Program. Office of Air Quality Planning and Standards. EPA-453/R-94-075. October.

USEPA. 1994b. Guidance For Assessing Chemical Contaminant Data for Use in Fish Advisories. Volume II: Risk Assessment and Fish Consumption Limits. Office of Water. Washington DC. EPA 823-B-94-004.

USEPA. 1995a. 1993 Toxics Release Inventory. USEPA, Office of Pollution Prevention and Toxics. EPA 745-R-95-010.

USEPA. 1995b. Great Lakes Water Quality Initiative Technical Support Document for the Procedure to Determine Bioaccumulation Factors. EPA-820-B-95-005. U.S. EPA, Office of Water, Washington, DC.

USEPA. 1996a. Proposed Guidelines for Carcinogen Risk Assessment (61 FR 17960, April 23, 1996).

USEPA. 1996b. USEPA's National Toxics Inventory Database Version 2.1. Prepared for USEPA by Radian Corporation. Draft.

USEPA. 1997. Carcinogenicity Risk Assessment for Lifetime Exposure. 1,3-Dichloropropene. verified August 4, 1993, minor correction of the number made in May, 1996. Unpublished. Available in the Public Docket for the Proposed Revisions to the Ambient Water Quality Criteria Human Health Methodology.

USEPA. 1998a. Federal Register Notice: Draft Revisions to the Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health.

USEPA. 1998b. Ambient Water Quality Criteria Derivation Methodology; Human Health. Technical Support Document. EPA/822/B-98/005. July.

USEPA. 1998c. Daily Average Per Capita Fish Consumption Estimates Based on the Combined USDA 1989, 1990, 1991 Continuing Survey of Food Intakes by Individuals (CSFII). Volume I: Uncooked Fish Consumption National Estimates; Volume II: As Consumed Fish Consumption National Estimates. Prepared by SAIC under Contract #68-C4-0046. March.

USFDA (U.S. Food and Drug Administration). 1990. Food and Drug Administration Pesticide Program: Residues in foods - 1989. Washington, DC. Journal of Assoc. Off. Anal. Chem. 73(5): 127A-146A.

USFDA (U.S. Food and Drug Administration). 1988. Food and Drug Administration Pesticide Program: Residues in foods - 1989. Washington, DC. Journal of Assoc. Off. Anal. Chem. 71(6): 156A-174A.

Valencia, R., J.M. Mason, R.C. Woodruff, and S. Zimmering. 1985. Chemical mutagenesis testing in Drosophila. III. Results of 48 coded compounds tested for the National Toxicology Program. Environ. Mutagen. 7(30): 325-348.

Van Duuren, B.L., B.M. Goldschmidt, G. Loewengart, and A.C. Smith. 1979. Carcinogenicity of halogenate olefinic and aliphatic hydrocarbons in mice. J. Natl. Cancer Inst. 63(6): 1433-1439.

Venable, J.R., C.D. McClimans, R.E. Flake, and D.B. Dimick. 1980. A fertility study of male employees engaged in the manufacture of glycerine. J. Occup. Med. 22(2):87-91.

Vithayathil, A.J., C. McClure, and J.W. Myers. 1983. Salmonella/microsome multiple indicator mutagenicity test. Mutat. Res. 121(1): 33-37.

Waechter, J.M., Jr. and P.E. Kastl. 1988. 1,3-Dichloropropene pharmacokinetics and metabolism in Fischer 344 rats following repeated oral administration. Unpublished study submitted by Dow Chemical USA, Midland, MI under TSCA 8d. OTS0516660.